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CRRT Experience in a patient diagnosed with anthracofibrosis in intensive care : a case report

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ABSTRACT

Bronchial anthracofibrosis is characterized by dark pigmentation (anthracosis) in the bronchial mucosa, often accompanied by bronchial stenosis or obstruction due to fibrosis in the same area. Diagnosis is typically established via bronchoscopy in patients who do not fully respond to treatment, exhibit radiological abnormalities, and have obstructive lung disease. Anthracofibrosis is mostly observed in conjunction with tuberculosis but can also frequently accompany pneumonia, necessitating close monitoring. In the intensive care unit (ICU), continuous renal replacement therapy (CRRT) is commonly used for critically ill patients with acute kidney injury (AKI) secondary to sepsis. It is particularly preferred in patients with acute renal failure (ARF) who have hemodynamic instability and are on inotropic agents. We present a case of a 78-year-old male admitted to the ICU due to pneumonia secondary to sepsis, septic shock, and AKI. During his ICU stay, frequent airway obstructions necessitated bronchoscopy, revealing a diagnosis of anthracofibrosis. CRRT was initiated due to hypotension and anuria under inotropic support. However, the patient exhibited disseminated intravascular coagulation (DIC) due to sepsis, along with intrabronchial hemorrhages and clot formations, resulting in airway obstruction and increased airway pressure. These complications hindered prolonged CRRT due to increased hemodynamic instability.

This case report aims to highlight the impact of increased airway pressure on CRRT in patients with obstructive lung diseases like anthracofibrosis. In such cases, the potential for increased hemodynamic instability should be considered, and measures to reduce airway pressure should be implemented.

Keywords: Anthracosis, airway pressure, septic shock, continuous renal replacement therapy

INTRODUCTION

Bronchial anthracofibrosis is characterized by dark pigmentation (anthracosis) in the bronchial mucosa, often accompanied by bronchial stenosis or fibrosis-related obstruction in the same area.[1] Diagnosis is commonly established through bronchoscopy in patients with obstructive lung diseases who exhibit radiological abnormalities and do not fully respond to treatment.[2] While the main cause of anthracosis/anthracofibrosis remains unknown, it is thought that exposure to biomass, genetics, chronic inflammatory reactions, air pollution, domestic pollution, and prolonged contact with chronic infections like tuberculosis play roles in the development of the disease.[3,4] While a review of the literature on anthracofibrosis shows it predominantly appears in conjunction with tuberculosis[1,5,6] it can also frequently coincide with pneumonia, necessitating close monitoring.[7,8]

In the intensive care unit (ICU), the incidence of Acute Kidney Injury (AKI) due to sepsis ranges from 15-20%, with CRRT (Continuous renal replacement therapy) being a frequently used treatment modality for critically ill patients.[9] Compared to intermittent hemodialysis, CRRT is preferred for its lesser hemodynamic instability and ability to be administered without anticoagulants or with alternative anticoagulation protocols in patients with bleeding disorders.[10]

Increased airway pressure in obstructive lung diseases can lead to hemodynamic disturbances.[11] In the case of septic shock, hemodynamic disturbance occurs, which leads to impaired tissue perfusion.[12] Although CRRT is a preferred dialysis method in hemodynamic disorders, its use is limited in severely hypotensive patients. Hypoxia and vasopressor use are independent risk factors for early mortality during CRRT.[13] This case report aims to draw attention to the hemodynamic disturbances resulting from airway obstruction in a patient with anthracofibrosis, emphasizing the challenges this poses for critical applications such as CRRT.

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CASE

A 78-year-old male presented to the emergency room with complaints of shortness of breath and altered consciousness. He was admitted to the ICU with diagnoses of pneumonia, sepsis, respiratory failure, and AKI. Thoracic (computed tomography) CT imaging showed findings consistent with infiltrative pneumonia, while brain CT and diffusion MRI (magnetic resonance imaging). revealed no abnormalities. Blood tests confirmed AKI and sepsis (Table). The patient's history included diabetes mellitus, hypertension, and a past cerebrovascular event. Initially, the patient received oxygen support via a reservoir mask but was electively intubated on the second day due to worsening respiratory distress and tachypnea, and switched to SIMV-VC mode. The cultures taken on the first day showed no growth, and the patient's cultures were sent again on the second day due to the development of fever.

The cultures taken on the first day showed no growth, but repeat cultures on the second day revealed fever development. Noradrenaline infusion was initiated due to hypotension. Fluid replacement therapy was administered based on hemodynamic monitoring. The patient was started on piperacillin/tazobactam and clarithromycin. Despite initial urine output of 1800cc and an intake and output (I&O) balance of 420, urine output decreased to 220cc on the second day, prompting a loop diuretic infusion. As the patient became anuric and remained on noradrenaline, CRRT was initiated but had to be terminated after 48 hours due to low venous return pressure alarms. Following the initiation of CRRT, the patient's urine output increased. During this period, the mechanical ventilator alarmed twice due to increased pressure and blockage in the endotracheal tube, necessitating its replacement. Clots were observed within the intubation tube. A fiberoptic bronchoscopy (FOB) was performed after the third blockage, revealing organized clots completely occluding the lumen from the mid-trachea downward. These clots could not be cleared via FOB and were subsequently removed using rigid bronchoscopy with basket and forceps biopsies. The clots extended distally into both main bronchi up to the segmental orifices, which were thoroughly cleaned. Attempts to recommence CRRT for the oliguric patient on high-dose inotropic support were unsuccessful, as it was terminated after two hours due to low-pressure alarms. The patient, who had melena during the night observations, had oral intake stopped, and a proton pump inhibitor infusion was administered. The melena continued for 2 days. Laboratory results (Table 1) and clinical findings suggested the presence of disseminated intravascular coagulation (DIC) secondary to sepsis.

Given the recurrence of clotting and hemorrhagic elements in the intubation tube and persistent pressure alarms on the mechanical ventilator, another FOB was conducted. This examination revealed anthracotic plaques covering all lobes and segments, with bronchi and segments obstructed by fibrin plugs, leading to a diagnosis of bronchial anthracofibrosis. During this period, the patient's blood culture grew methicillin-resistant staphylococcus Aureus (MRSA), and urine culture grew non-albicans Candida, necessitating a switch to tigecycline, fluconazole, and imipenem. Subsequent cultures showed no growth.

Table . Laboratory findings observed during the patient's intensive care admission

Blood values	Admission values	Post-first CRRT values	Post-first CRRT values	Post-fOB values	Post-IHD values
Leukocyte (K/ ul)	9.9	8.8	16.1	10.7	2.6
Hemoglobin (g/dl)	15.6	13.9	11.3	8.5	8.0
Platelets (cell x10⁹/L)	137.000	120.000	151.000	78.000	36.000
Urea (mg/dl)	134	268	99	404	252
Creatinine (mg/dl)	2.92	8.21	2.89	7.6	5.44
GFR (ml/dk)	21.3	6.2	21.5	6.7	10.0
Lactate (mmol/L)	4	3.5	2	1.8	3.8
CRP (mg/L)	401	261	247	127	179
PCT (ng/ml)	92.36	55.89	25.6	10.8	3.43
D-dimer (ng/ml)	25.290	35.200	14.280	31.600	14.810
aPTT (s)	34.2	32.8	37.4	31.2	42.4
PT (s)	16.8	18.2	16.4	21.4	19.9
INR	1.48	1.62	1.44	1.97	1.73

CRRT: Continuous renal replacement therapy, FOB: Fiberoptic bronchoscopy, IHD: Intermittent hemodialysis, GFR: Glomerular filtration rate, CRP: C-reactive protein, PCT: Procalcitonin, aPTT: Activated partial thromboplastin time, PT: Prothrombin time, INR: International normalized ratio

The patient's third CRRT attempt lasted seven hours before being terminated due to similar complications. Consequently, the patient underwent hemodialysis without heparin for two hours, during which one unit of erythrocyte suspension was transfused. CRRT or intermittent hemodialysis could not be continued due to the patient's hemodynamic instability while anuric and receiving high-dose inotropic support.

The patient's hemodynamic status and overall condition progressively deteriorated, culminating in cardiac arrest on the 20th day of ICU admission. Resuscitation efforts were unsuccessful, and the patient was pronounced deceased.

DISCUSSION

Anthracofibrosis is characterized as an obstructive lung disease marked by black pigmentation, known as anthracosis, and fibrosis in the bronchial mucosa, resulting in a narrowing that can obstruct the bronchial lumen. Increased airway pressure may also be observed due to airway obstruction.[2] In our patient, the mechanical ventilator frequently alarmed due to increased airway pressure. Despite changing the endotracheal tube, high airway pressure persisted, prompting a bronchoscopy that confirmed the diagnosis of anthracofibrosis. Bronchoscopy revealed fibrin plugs and clots, which were removed in an attempt to reduce airway pressure. Hyperinflation in patients with airway obstruction can disrupt venous return, potentially leading to hypotension and hemodynamic collapse.[14]

Hypotension is a significant risk factor for in-hospital mortality and can frequently occur in patients undergoing CRRT.[15] An increase in airway pressure in patients undergoing CRRT has been found to be an independent risk factor for mortality.[16] Our patient also had a sepsis and septic shock secondary to pneumonia. The patient was hypotensive due to both high airway pressure and septic shock. CRRT was applied due to the patient receiving inotropic support, being anuric, and exhibiting signs of fluid overload.

During CRRT, pressure alarms can occur due to various reasons, including blockages, hematomas in the vein, circuit obstruction or kinking, venous stenosis, high coagulation states, and hypotension. In our case, upon encountering a pressure alarm during CRRT, the central dialysis catheter's location was adjusted, ensuring smooth blood inflow and

outflow with no blockage in the CRRT line. The pressure alarm was attributed to hypotension caused by septic shock, DIC, and increased airway pressure. DIC is a systemic process causing both thrombosis and bleeding, and it is a frequent and serious complication of sepsis and septic shock, associated with high mortality and morbidity.[17] In our patient's follow-ups, DIC developed due to sepsis, leading to both gastrointestinal and intrabronchial bleedings. The resulting intrabronchial bleedings and clots also caused airway obstruction and increased airway pressure, in addition to anthracofibrosis.

The CRRT circuit can lead to the consumption of clotting factors and can increase the risk of clotting due to high pressure and turbulence in the connections. While systemic anticoagulation is often deemed sufficient to prevent the clotting of the recurring CRRT circuit in most cases, additional anticoagulant might need to be added in some situations.[18] Thrombocytopenia is commonly seen in patients undergoing CRRT, and it has been found that mortality is higher in patients who are thrombocytopenic and undergoing CRRT9.[19,20] In our case, even though the heparin dose was adjusted according to the aPTT value, most of the time, there was no need for heparin due to thrombocytopenia and increased aPTT resulting from DIC.

This study has limitations, it is based on a single case. Additionally, the patient's anamnesis lacked data regarding the etiology of anthracofibrosis, making it difficult to ascertain the exact cause of anthracofibrosis development. It is suspected that pneumonia may have been a contributing factor.

CONCLUSION

In cases of obstructive lung diseases such as anthracofibrosis, which do not respond to bronchodilators, it is crucial to recognize that increased airway pressure can significantly exacerbate hemodynamic instability in critically ill patients. Measures should be taken to reduce airway pressure to mitigate these effects.

ETHICAL DECLARATIONS

Informed Consent
The patient signed and free and informed consent form.

Referee Evaluation Process
Externally peer-reviewed.

Conflict of Interest Statement
The authors have no conflicts of interest to declare.

Financial Disclosure
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Author Contributions
All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

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REFERENCES

1. Bekci TT, Maden E, Emre L. Bronchial anthracofibrosis case with endobronchial tuberculosis. *Int J Med Sci*. 2011;8(1):84-87.
2. Kim YJ, Jung CY, Shin HW, Lee BK. Biomass smoke induced bronchial an- thracofibrosis: presenting features and clinical course. *Respir Med*. 2009;103(5):757-765.
3. Jamaati H, Sharifi A, Mirenayat MS, et al. What do we know about anthracofibrosis? A literature review. *Tanaffos*. 2017;16(3):175-189.
4. Yılmazel Uçar E, Araz Ö, Akgün M, et al. Bronşiyal antrakozis-antrakofibrozis: potansiyel nedenler ve klinik özellikler. *Eurasian J Pulmonol*. 2014;16(1):17-20.
5. Wynn GJ, Turkington PM, O'Driscoll BR. Anthracofibrosis, bronchial stenosis with overlying anthracotic mucosa: possibly a new occupational lung disorder: a series of seven cases from one UK hospital. *Chest*. 2008; 134(5):1069-1073.
6. Hemmati SH, Shahriar M, Molaei NA. What causes anthracofibrosis? Either tuberculosis or smoke. Pakistan J Med Sci. 2008;24(3):395.
7. Lee HS, Maeng JH, Park PG, et al. Clinical features of simple bronchial anthracofibrosis which is not associated with tuberculosis. *Tuberc Respir Dis*. 2002;53(5):510-518.
8. Rangelov K, Sethi S. The first described case of occupational anthracofibrosis in the USA. *Case Rep Pulmonol. 2014;14(23):460594.*
9. Cho AY, Yoon HJ, Lee KY, Sun IO. Clinical characteristics of sepsis-induced acute kidney injury in patients undergoing continuous renal replacement therapy. *Renal fail*. 2018;40(1):403-409.
10. Morabito S, Pistolesi V, Cibelli L, Pierucci A. Continuous renal replacement therapies (CRRT) will remain the most widely adopted dialysis modality in the critically ill. *G Ital Nefrol*. 2009;26(1):13-21.
11. Güven M. Mekanik ventilasyon uygulamaları. 11. Ulusal İç Hastalıkları Kongresi, 30 Eylül-4 Ekim 2009, Antalya, Kongre Kitapçığı. 52-56.
12. Yıldız F, Karakoç E. Sepsiste hemodinamik destek. *Arşiv Kaynak Tarama Derg*. 2014;23(2):157-167.
13. Prasad B, Urbanski M, Ferguson TW, Karreman E, Tangri N. Early mortality on continuous renal replacement therapy (CRRT): the prairie CRRT study. *Can J Kidney Health Dis*. 2016;36(3):124.
14. Gladwin MT, Pierson DJ. Mechanical ventilation of the patient with severe chronic obstructive pulmonary disease. *Intensive Care Med*. 1998;24:898–910.
15. Shawwa K, Kompotiatis P, Jentzer JC, et al. Hypotension within one-hour from starting CRRT is associated with in-hospital mortality. *J Crit Care*. 2019;54(1):7-13.
16. Flores FX, Brophy PD, Symons JM, et al. Continuous renal replacement therapy (CRRT) after stem cell transplantation. A report from the prospective pediatric CRRT registry group. *Pediatric Nephrology*. 2008; 23:625-630.
17. Tagami T, Matsui H, Horiguchi H, et al. Antithrombin and mortality in severe pneumonia patients with sepsis-associated disseminated intravascular coagulation: an observational nationwide study. *J Thromb Haemost*. 2014;12(9):1470-1479.
18. Foti L, Villa G, Romagnoli S, Ricci Z. Acute kidney injury and extracorporeal membrane oxygenation: review on multiple organ support options. *Int J Nephrol Renovasc Dis*. 2021;13(14):321-29.
19. Guru PK, Singh TD, Akhoundi A, Kashani KB. Association of thrombocytopenia and mortality in critically ill patients on continuous renal replacement therapy. *Nephron*. 2016;133(3):175-182.
20. Wu B, Gong D, Xu B, et al. Decreased platelet count in patients receiving continuous veno-venous hemofiltration: a single-center retrospective study. *PLoS One*. 2014;9(5):97286.